A STUDY OF 100 CASES OF LIVER ABSCESS

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF 100 CASES OF LIVER ABSCESS"** submitted by **Dr. K. Madhu suthan** to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai,** is in partial fulfillment of the requirement for the award of M.S. degree Branch I (General Surgery) and is a bona fide research work carried out by him under direct supervision and guidance.

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DECLARATION

I, **Dr. K. Madhu suthan ,** declare that I carried out this work on **"A STUDY OF 100 CASES OF LIVER ABSCESS "** at the Department of General Surgery, Government Rajaji Hospital during the period of December 2005 – June 2006. I also declare this bona fide work or any part of this work was not submitted by me or any other for any award, degree or diploma to any university, board either in India or abroad.

This is submitted to the **Tamil Nadu Dr. M .G .R. Medical University, Chennai** in partial fulfillment of the rules and regulation for the M.S. Degree examination in General Surgery.

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INTRODUCTION

LIVER ABSCESS is an entity that continues to pose diagnostic and therapeutic problems. Two major types of liver abscess are amoebic and pyogenic. Others include fungal and TB abscess that are observed more commonly in immunocompromised individuals.

In many countries amoebiasis and amoebic dysentery continue to be a common health problem. Amoebic infections of the liver, colon and other organs are common in areas of poor sanitation and particularly in developing nations with contaminated water supplies and poor public hygiene.

Amoebic liver abscess is aptly called as “Tropical liver abscess” as it is more common in tropics. Typically the endemic areas of amoebiasis are located in tropical and subtropical countries. The early description came from India only.

Careful history and skillful clinical evaluation may provide important information about the diagnosis. USG abdomen and serological tests are the most important investigations in the diagnosis of liver abscess

Entamoeba hisytolytica infection affects 10% of world population and sex distribution is 20:1 m:f ratio. Pyogenic liver abscess affects 30/100000 hospital admissions and the sex incidence is almost equal.

The management of amoebic liver abscess evolved with recognition of colonic amoebiasis as the antecedent source of the liver abscess. Early treatment with open surgical drainage alone had met with limited success. Efforts to treat both the liver abscess and the colonic infestation improved the success rate.

Later the development of systemic amoebicidal agents coupled with closed aspiration became the treatment of choice. The advent of laparoscopy has indeed eliminated the need for open drainage.

This study is mainly focused on the incidence and various modalities of presentation of liver abscess and the various modalities of treatment and their indications.

AIM OF STUDY

1. To study the epidemiological aspects of amoebic and pyogenic liver abscess.

2. To study the various modalities of clinical presentation.

3. To study the various modalities of treatment and their effectiveness in the treatment of liver abscess.

SURGICAL ANATOMY

EMBRYOLOGY[1,20]

The liver primodium appears in the middle of the third week as an outgrowth of the endodermal epithelium at the distal end of the foregut. This bud grows into the ventral mesogastrium and passes through it into the septum transversum.

It enlarges, soon shows a division into larger cranial part called the pars hepatica and the smaller caudal portion called the pars cystica. The pars hepatica divides into right and left parts each of which forms one lobe of liver.

The cells arising from this division are broken up into interlacing columnar cells called hepatic trabeculae. In this process, the umbilical vein and the vitelline vein are broken up to form the sinusoids of the liver. Sinusoids are also formed from the mesenchyme of the septum transversum.

The endodermal cells of the hepatic bud gives rise to the parenchyma of the liver and the bile capillaries. The mesoderm of the septum transeversum forms the capsule and fibrous tissue basis of the liver.

ANATOMY[4,5]

The liver the largest gland in the body weighs approximately 1500g and receives about 1500ml of blood /min. This wedge shaped organ occupies most of the right hypochondrium and epigastrium. It has two surfaces, visceral and diaphragmatic.

The diaphragmatic surface, convex, is divided into anterior, posterior and right surfaces. Sharp inferior border separates right and anterior surface from visceral surface.

The main vessels and ducts enter or leave at the porta hepatis which is on the visceral surface, but hepatic vein emerges from the diaphragmatic surface.

The inferior border is notched by the ligamentum teres. The falciform ligament ascends on the anterior surface to reach the superior surface where a reduplication of the left leaf forms the left triangular ligament. The right leaf becomes the upper layer of the coronary ligament.

The venacava lies in the deep groove on the posterior surface. To the right is the triangular bare area, with the vena cava as its base and with sides formed by superior and inferior layers of coronary ligament. The apex where these two layers meet is the right triangular ligament.

At the porta hepatic lie the hepatic ducts, hepatic artery and portal vein. They lie in the order -vein-artery-duct, with the duct in front. There are also nodes and nerves of the liver. The bare area is in contact with the diaphragm and right suprarenal gland.

The visceral surface is related to stomach, duodenum, hepatic flexure of colon and right kidney.

LOBES

Anatomically the liver has two lobes, right and left divided by the falciform ligament anteriorly. The right lobe is further divided into right lobe proper, caudate and quadrate lobes. The caudate lobe lies in between IVC and fissure for ligamentum venosum. The quadrate lobe lies in the visceral surface between the gallbladder fossa and fissure for ligamentum teres.

SURGICAL LOBE

The liver is divided into right and left lobe equally by line through the bed of the gallbladder towards the IVC. This is the cantlie's line.

SEGMENTS OF LIVER

On the basis of blood supply and biliary drainage there are four main hepatic sectors: left lateral, left medial, right anterior and

right posterior .These four sectors are further subdivided into eight segments.

SEGMENT 1- CAUDATE LOBE - An autonomous segment receiving blood from right and left branches of hepatic artery and portal vein, draining bile into right and left hepatic ducts and having independent venous drainage into IVC.

The left lateral sector contains segment II posteriorly, segment III anteriorly with the left hepatic vein between them. Segment IV is recognized on the visceral surface as the quadratelobe. Segment V and VI are the inferior segments of the right anterior and posterior sectors respectively. Segments VII and VIII are the superior segments of the right posterior and anterior sectors respectively.

HISTOLOGY[20]

Liver is seen to be composed of parenchymal cells arranged in anastomosing and branching plates which form a three

dimensional lattice. Plates of parenchymal cells radiate from central vein like spokes of a wheel, each being 1mm in diameter. Portal triad or portal areas or portal canal contains a branch of portal vein, a branch of hepatic artery and an inter lobular bile ductule.

In humans, liver contains 3-6 portal canals per lobule. Between parenchymal plates are sinusoidal blood spaces. Sinusoids are irregularly disposed, normally in a direction perpendicular to the lines connecting central veins. Walls of the sinusoids consists of endothelial cells called Kupffer cells. Potential spaces between hepatic cells and walls of sinusoids are called space of Disse. This space is continuous with larger space that surrounds the portal areas known as the space of Moll.

The continuous liver tissue is pervaded by two systems of tunnels, the portal tracts and hepatic canals, which are arranged in such a way that they do not meet each other. As far as possible two

systems run in planes perpendicular to each other. The terminal branches of portal vein discharge their blood into sinusoids .

REVIEW OF LITERATURE

AMOEBIC LIVER ABSCESS

INCIDENCE[16,7,18]

It has the highest incidence in tropical and sub-tropical countries and in areas with poor sanitation. Hepatic abscess occurs in 3 to 7% of all patients with amoebiasis.

Mean age of the patient is 30 to 40.

Male to female ratio is 9:1

HISTORY

In 1875, Losch discovered Entamoeba histolytica as the causative factor for amoebic dysentery.

In 1890, Sirosler first reported the presence of amoeba in a liver abscess as well as in the stool of the same patient.

In 1891, Councilman and Lafluer first used the term amoebic liver abscess. In 1992, Rogers demonstrated that active

amoeba were present infrequently in the pus but were found in the wall of the abscess.

ETIOPATHOGENESIS[17,20]

The cystic form of the E.histolytica is the infecting agent, usually through contaminated water. They are resistant to acid PH and drying, digested by trypsin in the small intestine and four invasive trophozoites are released, which multiply in the caecum.

Trophozoites exist in two forms 1) small 2) large. When tissue invasion occurs trophozoites ingest RBCs and become large forms. The most frequent site of extraintestinal colonization is liver. The three possible routes are 1) portal vein 2) lymphatics 3) direct extention, Most frequently through portal vein. In liver trophozoites lodge in small vessels and produce thrombosis and infarction of small areas of hepatic parenchyma. From there, it releases enzyme to produce cytolysis. This stage is called amoebic hepatitis.

Healing at this stage or further progression is decided by host's immune status and host's nutritional status. If it progresses, coalition of a number of small areas of necrosis produce a macroabscess.

The pus is dark reddish in colour (ANCHOVY SAUCE) sterile, consists of mixture of blood and destroyed liver tissue. Trophozoites are rarely found in the pus but present in the wall of the cavity.

Abscess cavity varies in size from 1 to 25 cm. Leukocytic infiltration and inflammatory reaction are characteristically absent. If untreated will rupture into adjacent organs like peritoneum, pleural cavity and pericardium. It is more likely to be solitary and located in right lobe in the posterosuperior aspect.

HOST FACTORS

HUMAN HOST REPRESENTS THE MAJOR RESERVOIR.

Interperson transmission occurs via flies and food handlers and by sewage contamination of water sources. Male homosexuals also transmit the disease.

Menstruating women and breast fed children have low incidence of invasive infection due to presence of protective IgA in the mother's milk and to the low iron content of the milk. High content of iron in the diet, high carbohydrate, patient with decreased immunity are predisposing factors to invasive amoebiasis.

SYMPTOMS AND SIGNS[3,4]

It may present as acute inflammatory process or chronic indolent disease. Acute presentations are more common. Rarely an individual with a ruptured liver abscess may present with shock.

Interval between amoebic dysentery and development of liver abscess was around 2 months in 505 cases but it may occur after years.

Symptoms:

Most common is pain

Fever/nausea /vomiting/cough /pleurisy/

Diarrhea

Jaundice

Signs

Rt. upper quadrant tenderness

Hepatomegaly

Fever/jaundice

Pleural effusion

INVESTIGATIONS

1) STOOL EXAMINATION

2) SEROLOGICAL TESTS

3) ULTRASOUND

4) CT/MRI

5) SCINTIGRAPHY

ST00L EXAMINATION

The reported incidence of finding amoebic cysts or trophozoites in the stool of patients with amoebic liver abscess varies considerably.

Previous studies found amoeba in the stool in less than 15% ALA patients.

Fresh stool specimen must be preserved in formalin or polyvinyl alcohol and can be stained in buffered methylene blue, trichlorane or iodine.

SEROLOGICAL TESTS[1,18]

These procedures are necessary to confirm the presence of amoebic liver abscess. It should be obtained as soon as the diagnosis of a liver abscess is entertained.

Indirect haemagglutination test (IHA), gel diffusion precipitation(GDP), complement fixation, latex agglutination, countercurrent immunoelectrophoresis, cellulose acetate precipitin, ELISA and identification of a recombinant protein are the tests available.

If positive these tests indicate current or previous amoebic infection. The IHA, GDP have been the most frequently used serological tests. IHA will remain positive frequently for many years after invasive amoebiasis, whereas the GDP was negative in 6 months. Advantages of GDP are simple to perform, inexpensive and provide information in 24 to 48 hrs.

The most recently developed test measures 29kpa peripheral membrane protein of pathogenic E.histolytica. It differentiates pathogenic from nonpathogenic strains and is highly specific and reasonably sensitive.

LABORATORY DATA(19)

Leucocytosis is seen in 70% of patients. Around 50% have elevated alkaline phosphatase and anemia. Serum bilirubin, SGOT, SGPT are mildly elevated. The presence of jaundice is associated with higher incidence of complications and a higher mortality rate.

IMAGING STUDIES(13)

X-ray –About two third of the patients have an abnormal chest radiograph. The most common radiological finding is elevated right hemidiaphragm, Rt. pleural effusion, Rt. lower lobe infiltration and hepatomegaly found less frequently. Fluoroscopy of the diaphragm reveals decreased motion.

Ultrasound– Helps in determining number, size and location of the abscess and can be used as a guide for percutaneous aspiration. Because USG is noninvasive, rapid,

relatively inexpensive and reproducible, it is also ideal for follow up.

Diagnostic accuracy-90%

BOULT BEE - sonographic feature of amoebic liver abscess

1. Smooth wall-68%
2. Less dense internal echoes compared to surrounding Liver-84%
3. Decrease in echoes -98%

SCINTISCAN

GALLIUM SCANNING is used to differentiate amoebic liver abscess from pyogenic abscess. Amoebic abscess will demonstrate a peripheral uptake around a central cold area whereas pyogenic abscess shows increased uptake of gallium throughout the abscess.

Amoebic liver abscess – cold spot

Pyogenic liver abscess - hot spot

CT AND ARTERIOGRAPHY

Smaller lesions can be detected and it is reserved for patients suspected of having an amoebic liver abscess in which ultrasound is not diagnostic.

Arteriography shows a non-vascular space occupying lesion. It is invasive and expensive and it may not be as accurate as USG. Better delineation of organs in the vicinity of liver is possible.

MRI

Amoebic abscess have multiple rims of variable signal intensity. Unfortunately bacterial abscess, intrahepatic hematoma and necrotic tumors also have similar characteristics. It may be useful for followup of treated cases and also in differentiating from a hepatic neoplasm.

TREATMENT

1) MEDICAL MANAGEMENT

2) SURGICAL MANAGEMENT

PERCUTANEOUS ASPIRATION

PERCUTANEOUS CATHETER DRAINAGE

OPEN SURGICAL DRAINAGE

LAPAROSCOPIC DRAINAGE

MEDICAL TREATMENT

DRUGS

BOTH LUMINAL AND TISSUE AMOEBICIDE

a) Metronidazole, Tinidazole, Secnidazole

b) Emetine, Dehydroemetine

LUMINAL AMOEBICIDE

a) Diloxanide furoate

b) Quiniodochlor, Diiodohydroxyquin

c) Tetracycline

TISSUE AMOEBICIDE

Chloroquine

A oral course of metronidazole 750mg 3 times a day for 10 days cures approximately 95% of patients with amoebic liver abscess.

INDICATIONS FOR CHLOROQUINE

a) To ensure no motile forms in liver

b) Resistant amoebiasis

For fulminant patients with pulmonary complications- Emetine, Dehydroemetine are useful. Cure rate is high if used for abscess less than 1.5 cm. It is also applied when abscesses are multiple or not amenable to aspiration.

PERCUTANEOUS ASPIRATION[15,20]

INDICATIONS

1) Therapeutic trial with antiamoebic drugs is deemed inappropriate.

2) When fever and pain persist for more than 3 to 5 days after starting appropriate therapy, aspiration may provide symptomatic relief.

3) In extremely large abscess where rupture is suspected to be imminent especially when pericardial rupture from a Lt. lobe abscess appears likely.

4) Diagnostic –when diagnosis remains uncertain despite serological and stool examination.

Those who argue against routine aspiration, say resolution time was unchanged by therapeutic aspiration and it increases the secondary infection rate.

Those who favour aspiration claim that the advantage of early resolution, preventing rupture and relieving pain out weigh the low incidence of secondary infection. Aspiration is also valuable when metronidazole therapy is contraindicated such as in pregnancy.

PERCUTANEOUS CATHETER DRAINAGE[15]

The reasons why PCD has not become popular include fear of bacterial superinfection and the fact that most patients respond to amoebicide therapy with or without closed aspiration.

In 1992, FLICE and his Italian colleagues employed intralesional chemotherapy, through PCD and documented fewer days of fever and a shorter hospital stay mainly for pulmonary /peritoneal complications.

Soft drainage catheters of sufficient size are needed to adequately drain thick, viscous contents of an amoebic abscess.

OPEN SURGICAL DRAINAGE[5,20]

Reserved for patients with complications.

1. Abscess that have failed to respond to more conservative therapy.

2. In the rare event of life threatening haemorrhage.

3. Abscess that erodes into the neighbouring viscus.

4. Patients with septicaemia from secondarily infected amoebic abscess.

LAPROSCOPIC DRAINAGE

It can be done in place of percutaneous drainage for lesions located anteriorly and superficially. For large lesions with impending rupture- avoids laparotomy

COMPLICATIONS[10]

The incidence of complications varies from 5 to 15%. The most common complications involve the pleura and the lung. Others are rupture into peritoneum, rupture into pericardium, secondary infection, bacteremia, amoebiasis cutis etc.

PERITONEAL AND VISCERAL INVOLVEMENT

Incidence of rupture varies between 2.5 to 15%.Rupture into hollow viscus like stomach, duodenum, colon can occur.

Free rupture is uncommon- occurs in nutritionally depleted and immunocompromised patients. Sudden bloody diarrhea, hematemesis may occur in colonic, gastric fistula.

USG and CT may show perihepatic fluid collection.

Laparotomy done, peritoneal wash given, tube drains placed and retained in the cavity. Hollow viscus perforation of abscess is dealt with exteriorization, proximal diversion, serosal patch closure and postoperative antiamoebic treatment.

THORACIC & PLEUROPULMONARY INVOLVEMENT

May be due to sympathetic effusion or rupture into pleural cavity. Patient may manifest with dyspnoea and dry cough, hypochondriac pain. Basal crepitations and pleural rub may be present. USG and CT often pickup pleural effusion. Rupture into bronchi may produce chocolate coloured sputum.

Treatment consists of thoracocentesis. ICD is to go high on the right lateral side of chest wall near axilla.

PERICARDIAL INVOLVEMENT

Abscess of Lt.lobe of liver are more prone to pericardial complications which may range from asymptomatic pericardial effusion to cardiac tamponade. In the presence of cardiac tamponade, aspiration of the pericardium must be performed along with the drainage of the liver abscess followed by antiamoebic drugs.

TREATMENT

Although investigators agree that all patients should be treated with amoebicidal drugs, some controversy persists with regard to choice of medication and the need for aspiration or surgical drainage.

PYOGENIC LIVER ABSCESS

Patients with pyogenic liver abscess are now more likely to be older, to be female and to have a biliary etiology. In 1836, John Bright provided the first description of the disease. In 1938, Ochsner associated appendicitis as the most common site of origin.

ETIOLOGY AND PATHOGENESIS[1,15]

Most PLA are due to infection in the biliary or intestinal tracts. Routes of infection are 1)Biliary 2)portal vein 3)hepatic artery 4)direct extension 5)traumatic 6)cryptogenic.

Biliary system is the common origin of PLA (35%). Presently, frequent sources of portal vein sepsis resulting in liver abscess include diverticulitis, perforated ulcers and perforated carcinoma. Appendicular, pelvic, pancreatic abscess are relatively rare sources.

“MCDONALD and HOWARD” noted that 65% are in right lobe, 12% in left lobe and 23% are bilateral.

The percentage of multiple abscesses varies between 30 and 70 and mortality correlates closely with the number of abscess. Gram negative aerobes were cultured in two thirds and gram positive aerobes in around 28% of patients .PLA is sterile in about 7% of patients.

ORGANISMS

The pyogenic nature of PLA is usually confirmed by microbiological culture. Positive abscess culture is found in approximately 80%, whereas blood cultures are positive in 60% of cases.

E.coli is the frequent organism. Others include klebsiella, enterococci, pseudomonas, citrobacter, proteus, Streptococci, staphylococci, etc. Hepatic abscess after trauma is caused by staphylococci, streptococci. In children with chronic granulomatous disease, hematological malignancy-staph aureus is the

predominant organism causing liver abscess. In few patients with AIDS- mycobacterium tuberculosis is a common infecting agent.

SYMPTOMS AND SIGNS

Most patients present with acute symptoms. Fever is the most common symptom. Others are right upper quadrant pain, malaise and nausea. The most common signs are enlarged liver 60% and jaundice 30%.

INVESTIGATIONS

LABORATORY DATA[19]

Leucocytosis and anemia are observed in 2/3rd of the patients. Elevated alkaline phosphatase and gamma glutamyl transpeptidase occurs in 965 of patients. Hyperbilirubinemia and hypoalbuminemia are associated with poor risk.

X-RAY:

Chest X-ray - Elevated Rt. hemidiaphragm, Rt.lower lobe,

Atelectasis, Rt.pleural effusion.

Abdomen - Hepatomegaly, gas within abscess

(erect)

USG:

Sensitivity reported upto 85 to 95%.Cannot always visualize liver dome. Fatty infiltration may produce markedly echogenic liver and multiple abscesses are difficult to detect. Has same resolution as scintiscan but has no radiation exposure. Can only identify >2cm in diameter. It is useful in differentiation of cystic and solid masses and in diagnosing the associated gall stones.

CT

Sensitivity upto 95%. It detects intrahepatic lesions upto 0.5cm. CT better delineates small abscess near the diaphragm and abscess in fatty livers. But it cannot always differentiate abscess from other SOL. Fungal hepatic abscess usually arise in patients

with leukemia, lymphoma and in patients with other malignancies receiving chemotherapy.

COMPLICATIONS

Complications occur in 45% of patients and were associated with significantly higher mortality. Pulmonary and pleural complications, septicemia, subphrenic or subhepatic abscess, rupture into peritoneum, rupture into pericardium and multiorgan failure are the complications encountered.

TREATMENT

ANTIBIOTICS

Early institution of antibiotic therapy is important. The antibiotic regimen is based on knowledge of spectrum of organisms isolated in PLA. The organisms cultured most frequently are

1.Gram negative aerobes

2. Streptococcal species

3. Anaerobes

Unless a specific bacteria have been isolated, a combination of penicillin, an aminoglycoside, an anaerobic spectrum is necessary. The duration of antibiotic therapy is individualized. Patients with multiple miliary abscesses require a shorter course. It is useful in multiple small abscesses that are not associated with abdominal disease that require surgery.

NEEDLE ASPIRATION

Useful in young, healthy patients with single large abscess and also diagnostic.

PERCUTANEOUS CATHETER DRAINAGE

Can be used in all cases of PLA along with antibiotics. Should not be used in Associated diseases that require open surgery, coagulopathy, anatomical inaccessibility, multiple miliary abscesses, ascites.

OPEN DRAINAGE[5,20]

It is indicated in PLA with intra abdominal disease requiring surgery and in areas inaccessible to PCD (ie close to portal vein). It may be through extraperitoneal or transperitoneal approach.

EXTRAPERITONEAL APPROACH

This is recommended for fear of peritoneal or pleural contamination. Here the abscess cavity is entered through an area of adhesion between the liver and the parietal peritoneum. The exact route was dictated by the position of the abscess and it should be as short as possible.

Disadvantages of this approach are that it does not allow adequate exploration of the entire liver or for recognition of an inadequately treated intraabdominal source of infection.

TRANSPERITONEAL APPROACH

This can be performed safely now with systemic antibiotic coverage and proper surgical technique. Its advantages are

1. Excellent exposure of the liver.

2. Best drainage site can be determined.

3. Multiple abscesses can be located.

4. If source of abscess is occult, entire abdomen can be explored.

5. If indicated CBD can be explored and drained.

If the abscess is not obvious during surgery intraoperative USG is used.

MATERIALS AND METHODS

Hundred cases of liver abscess were studied in Government Rajaji Hospital, Madurai Medical College, between June 2004 and June 2006.

All patients were thoroughly examined and case sheets were written in same set pattern to facilitate later comparison.

DIAGNOSTIC CRITERIA

All of them had several investigations required to approach the diagnosis and they were diagnosed as amoebic or pyogenic liver abscess. Basically USG abdomen, serology and pus c/s were done. Serology positive and USG characteristics of smooth wall, homogenous with no internal echoes and superficial solitary abscess were grouped as amoebic. Serology negative and pus c/s

negative cases with USG characteristics of amoebic abscess with were also considered as amoebic abscess .

LUNG INVOLVEMENT

X-ray chest PA view was taken in all cases. X-ray findings of rt. pleural effusion, rt. lower lobe infiltration in presence or absence of cough, expectoration were considered as positive lung involvement.

TREATMENT SELECTION

Cases with abscess cavity less than 5 cm were treated by drug therapy alone. Failure to relieve symptoms within 3 to 4 days were treated by percutaneous aspiration.

Those with abscess cavity greater than 5 cm were treated either by percutaneous aspiration or by percutaneous catheter drainage. Bilateral abscess cavities that were small and multiple were managed by medical therapy and when any one of the cavity is larger than 5 cm, it was managed by percutaneous aspiration.

Abscess cavities restricted to left lobe were treated by drug therapy if they were multiple, less than 5 cm and if greater than 5 cm and single were managed either by percutaneous aspiration or by laproscopic drainage.

Those abscess cavities that were larger than 10 cm or with chances of impending rupture, in segment III, IV, V, VI were managed by laproscopic drainage.

ADOPTED THERAPEUTIC PROTOCOL

MEDICAL

Abscess cavities that were less than 5 cm were treated by Tab. Metronidazole 750mg tds for 10 days.

Patient was on i.v metronidazole for three days initially or till the fever subsided, later oral metronidazole, percutaneous aspiration was done. continued. If patient had persisting symptoms after 3 to 4 days

PERCUTANEOUS ASPIRATION

Patient with abscess cavity > 5 cm were treated either by percutaneous aspiration or PCD. Multiple abscesses were percutaneously aspirated and also those failed with medical therapy.

Done by using 16G or 18G aspiration needle or 3 way adopter as a single prick under USG guidance. First, aspiration done followed by drugs. If symptoms are not decreasing after 3 days, do USG and assess the cavity size.

If the cavity is increasing in size or not decreasing do 2nd aspiration and continue drug therapy. Still the symptoms are not subsided by 7th postaspiration day and USG showed the cavity is not decreasing or increasing in size, consider PCD or laproscopic drainage.

PERCUTANEOUS CATHETER DRAINAGE

PCD was done by using Malecots /22 F foleys under ultrasound guidance with closed drainage system.

REMOVAL OF PCD

1. If the Quantity is less than 30ml /8hrs.

2. IF the drainage is not purulent.

3. USG and cavitogram were done to assess the cavity size. Note down the decrease in size of the cavity and the PCD can be removed.

LAPROSCOPIC DRAINAGE

Patients with large abscess greater than 10cm and large abscess that was located in the left lobe of liver not amenable to percutaneous drainage were treated by laproscopic catheter drainage. Smaller 16 /14 F foleys used for abscess drainage and the same criteria of removal similar to that of PCD was employed.

ANALYSIS OF DATA

Total patients studied—100

Amoebic liver abscess---90 (m-78, f-12)

Pyogenic liver abscess---10 (m-6, f-4)

Rt. lobe involved - 71

Lt. lobe involved - 13

Both lobes involved -16

SYMPTOM ANALYSIS

PAIN -84%

FEVER -77%

ANOREXIA _40%

JAUNDICE -5%

SIGNS

RT. HYPOCHONDRIAC PAIN -80%

HEPATOMEGALY -42%

TREATMENT ANALYSIS

ONLY MEDICAL - 30

DRUGS +PERCUTANEOUS ASPIRATION - 51

DRUGS +PERCUTANEUS CATHETER DRAINAGE - 11

DRUGS +LAPROSCOPIC CATHETER DRAINAGE - 8

PERCUTANEOUS ASPIRATION DONE AFTER FAILED drug therapy- 3

TABLE 1

AGE INCIDENCE

AGE	ALA	%	PLA	%
1-10				
10-20	5	5.5		
20-30	17	18.8		
30-40	38	42.2	1	10
40-50	22	24.4	2	20
50-60	8	8.8	5	50
60-70			2	20

TABLE 2

SEX INCIDENCE

TYPE	no .of patients	MALE	FEMALE	M:F
ALA	90	78	12	6.5:1
PLA	10	6	4	3:2

TABLE 3

COMPARISON OF NUMBER OF ABSCESS

S.NO	TYPE OF ABSCESS	ALA NO	PLA NO
1.	SINGLE	74	3
2.	MULTIPLE	16	7

TABLE 4

SYMPTOM AND SIGNS

SNO	SYPTOMS & SIGNS	ALA NO.	ALA %	PLA NO.	PLA %
1	PAIN	77	85.5	7	70
2	FEVER	69	76.6	8	80
3	NAUSEA/VOMITING	63	70%	7	70%
4	ANOREXIA	35	38.8	5	50
5	JAUNDICE	3	3.3	2	20
6	HEPATOMEGALY	35	38.8	7	70
7	® HYPOCHONDRIAC TENDERNESS	72	80	8	80
8	LUNG SIGNS	23	25.5	1	10

TABLE 5

COMPARISION OF TREATMENTS

NO	TREATMENT GROUP	RELIEF OF SYMPTOM	RESOLUTION TIME
1.	A)DRUG ALONE	7 DAYS	75 DAYS
2.	B)DRUG & ASPIRATION	4 DAYS	97 DAYS
3.	C)DRUG&PCD	3 DAYS	83DAYS
4.	D)DRUG&LCD	3 DAYS	85 DAYS

DISCUSSION

1. Amoebic liver abscess is common in 3rd to 4th decade whereas pyogenic liver abscess is common in 5th and 6th decade

2. M:F RATIO

Amoebic liver abscess - 8:1

Pyogenic liver abscess - 3:2

3. In this study most of the patients are labourers and of low socioeconomic status. It was also found to be common among alcoholics.

4. Most common symptom in amoebic liver abscess was pain (84%). It was followed by fever. In pyogenic liver abscess, the most common symptom was fever(80%).

5. Most common sign both in amoebic and pyogenic liver abscess was rt. hypochondriac tenderness(80%).

6. Hepatomegaly(70%) and anorexia(40%) are more common with pyogenic than amoebic liver abscess .

7. Lung signs characterised by X-ray findings, clinical symptoms were common with amoebic (25%) than pyogenic liver abscess(10%).

8. Serology (IHA) is important in the diagnosis of amoebic liver abscess. It is positive in 95% cases.

9. Jaundice was more common with pyogenic liver abscess (20%) than amoebic liver abscess.

10. Amoebic liver abscess was solitary in 82 % and multiple in 18% cases. It was restricted to rt. lobe in 74% and in 13% occupied left lobe and in 12% it was bilateral.

11. Pyogenic liver abscess was solitary in 30% and multiple in 70% cases. It was predominantly bilateral in 50% cases and was restricted to rt. lobe in 40% cases and left lobe in 10% cases.

12. Amoebic liver abscess mostly respond well to drug therapy alone. When used for abscess less than 5 cm there was relief of symptoms in 7 days on an average. The cavity resolved in an average of around 75 days. There were 3 cases of treatment failure that were later treated by percutaneous aspiration.

13. Both percutaneous aspiration and percutaneous catheter drainage when used for abscess cavity greater than 5 cm induced

early symptomatic relief (4 and 3 days respectively) and early resolution of abscess cavity with only (93 days and 82 days respectively) slight difference between the two.

14. Percutaneous aspiration was comparatively an easy procedure and can be repeated any number of times and avoids the risks and complication associated with a catheter in peritoneal cavity. It had better patient compliance than percutaneous catheter insertion

DISADVANTAGE: Repeated aspiration, follow up using ultrasound only, in very large abscess cavity, require repeated aspiration and not in cases with impending rupture.

15. Percutaneous catheter drainage induced rapid resolution of symptoms (3 days) and abscess cavity (82 days) but difficult in multiple liver abscesses. Follow up can be done both by using USG and cavitogram.

DISADVANTAGE: Requires extended period of hospital stay and i.v antibiotics due to catheter related infection. Patient compliance was poor due to presence of catheter in situ. Prolonged BT, CT is a contraindication.

16. Laproscopic liver drainage was employed for large abscesses, abscess with impending rupture and superficial abscesses in left lobe liver. Is less morbid than laparotomy.

DISADVANTAGE: General anaesthesia is necessary. Posterior segments (7/8) difficult to drain and periportal areas difficult to treat.

CONCLUSION

1. Amoebic liver abscess occurs in a younger age group (3rd & 4th decade) compared to pyogenic liver abscess (5th & 6th decade).

2. Both liver abscesses were common in men (8:1 in amoebic and 3:2 in pyogenic).

3. Pain is the most common symptom in amoebic liver abscess.

   Fever is the most common symptom in pyogenic liver abscess.

4. The most common symptom in both liver abscess was rt. hypochondriac tenderness.

5. Hepatomegaly, jaundice, anorexia were common with pyogenic liver abscess.

6. Lung involvement was common in amoebic liver abscess.

7. Drug therapy was effective when abscess cavity was smaller in size.

8. Percutaneous aspiration and percutaneous drainage have similar efficacy in early symptomatic relief and abscess resolution but percutaneous aspiration had better patient compliance.

9. Laproscopy avoided laparotomy for drainage of huge abscess with impending rupture and has similar symptomatic relief and resolution time compared to percutaneous catheter drainage with reduced morbidity and hospital stay.

ABBREVATIONS

ALA	:	amoebic liver abscess
PLA	:	pyogenic liver abscess
PCD	:	percutaneous catheter drainage
IHA	:	Indirect Haemagglutination test
GDP	:	gel diffusion precipitin
ELISA	:	Enzyme linked immuno sorbent assay
USG	:	ultrasound
CT	:	computed tomography
MRI	:	magnetic resonance imaging
AIDS	:	Acquired immuno deficiency syndrome
C/S	:	culture and sensitivity

PROFORMA

Case no : Ip no:

Name: age: sex: occupation:

Religion: DOA:

Address: DOD:

1. Complaints:

a) Pain abdomen- present/absent

-onset

-duration

-type

-site

-radiation

-relation to cough

-relation to food

b) Fever - high grade/low grade

- sustaining/spiking

c) Nausea/vomiting

d) Appetite and loss of weight

f) Jaundice

h) Cough- duration

dry or productive

sputum - amount & colour

4. Personal history

_ diet

- alcoholism

5. PHYSICAL EXAMINATION

General examination

Fever

Jaundice

6. VITALS

7. SYSTEMIC EXAMINATION

Abdomen— Hepatomegaly

Rt. hypochondriac tenderness

8. OTHER SYSTEMS EXAMINATION

RS: Rt. lower lobe crepitations (y/N)

Decreased breath sounds (y/N)

INVESTIGATIONS:

URINE- albumin/sugar/deposits

Blood routine- repeat USG

STOOL- ova/cysts/trophozoites cavitogram

Pus C/S

SEROLOGY (IHA)

LFT—

X-ray chest PA view resolution time

USG abdomen

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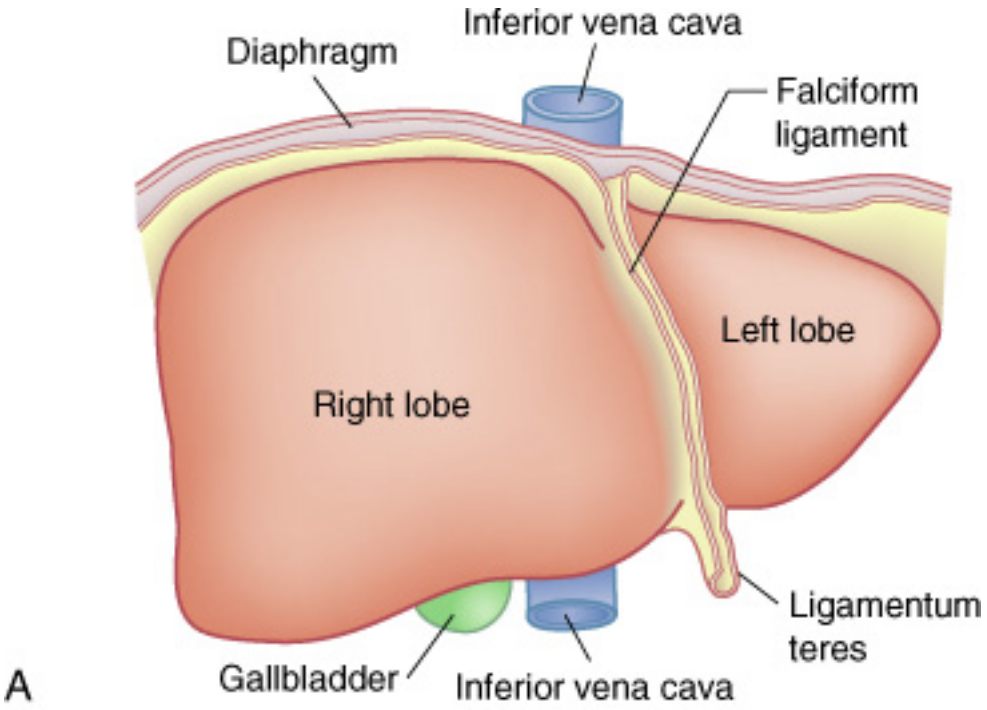
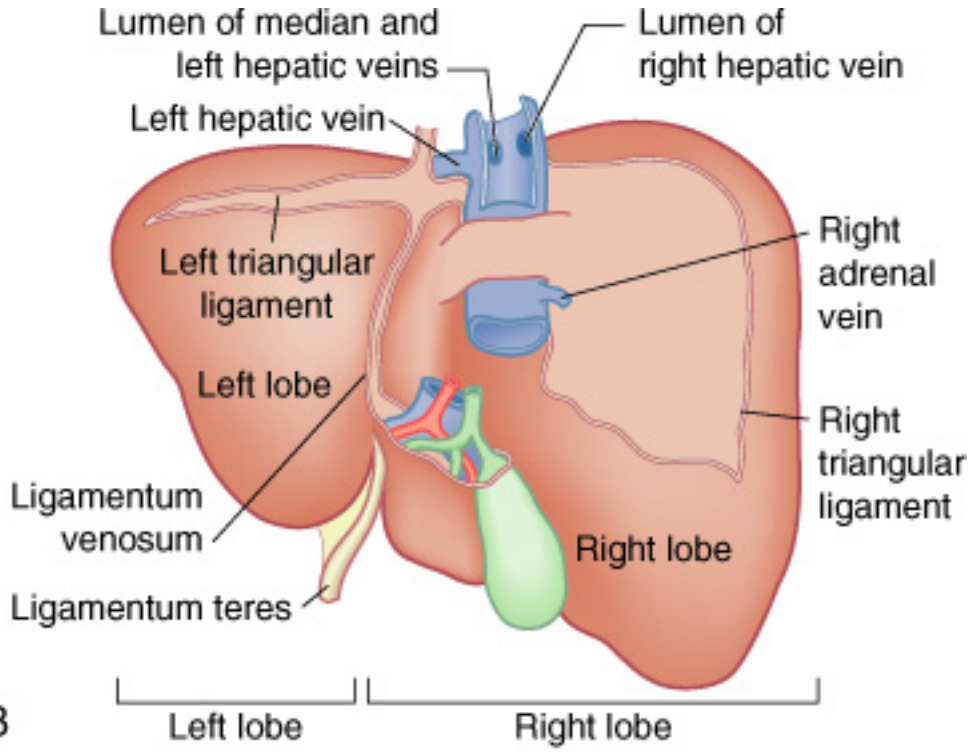
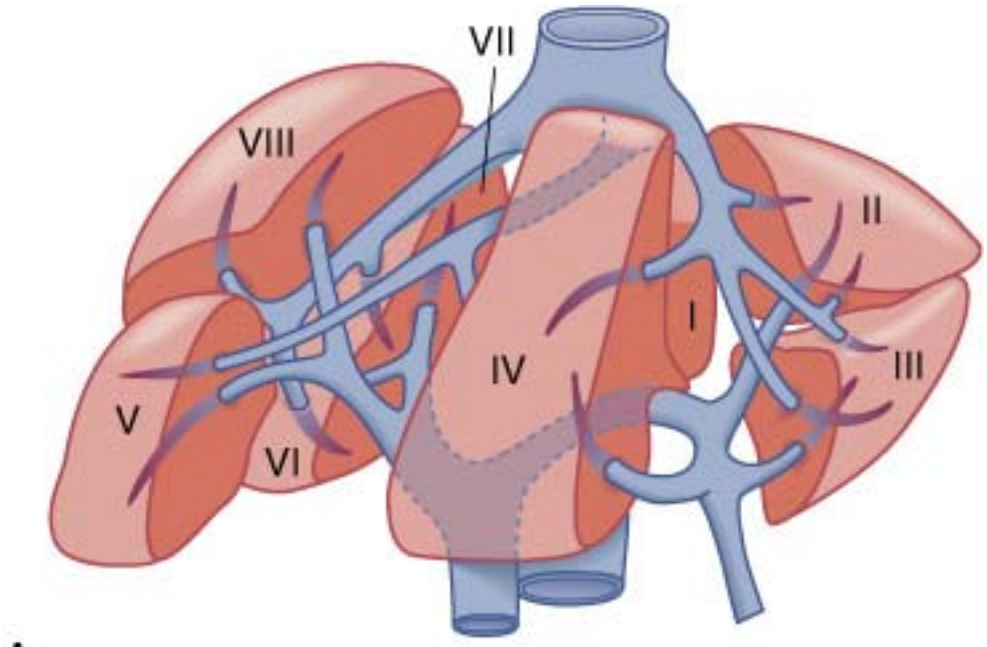

Diaphragm
Inferior vena cava
Falciform ligament
Left lobe
Right lobe
Ligamentum teres
Gallbladder
Inferior vena cava

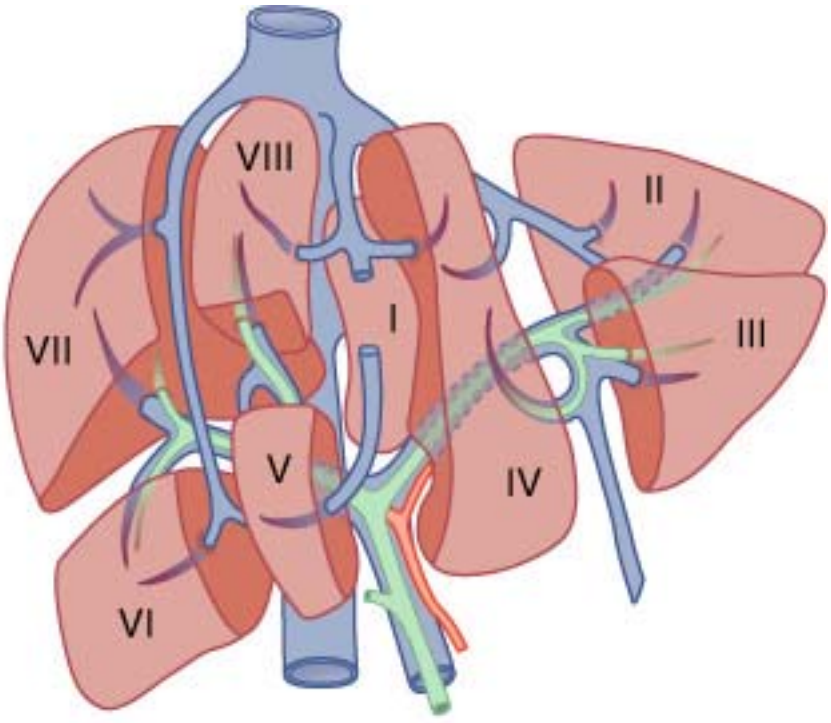
A


Lumen of median and left hepatic veins
Lumen of right hepatic vein
Left hepatic vein
Left triangular ligament
Right adrenal vein
Left lobe
Right triangular ligament
Ligamentum venosum
Right lobe
Ligamentum teres
Left lobe
Right lobe

B

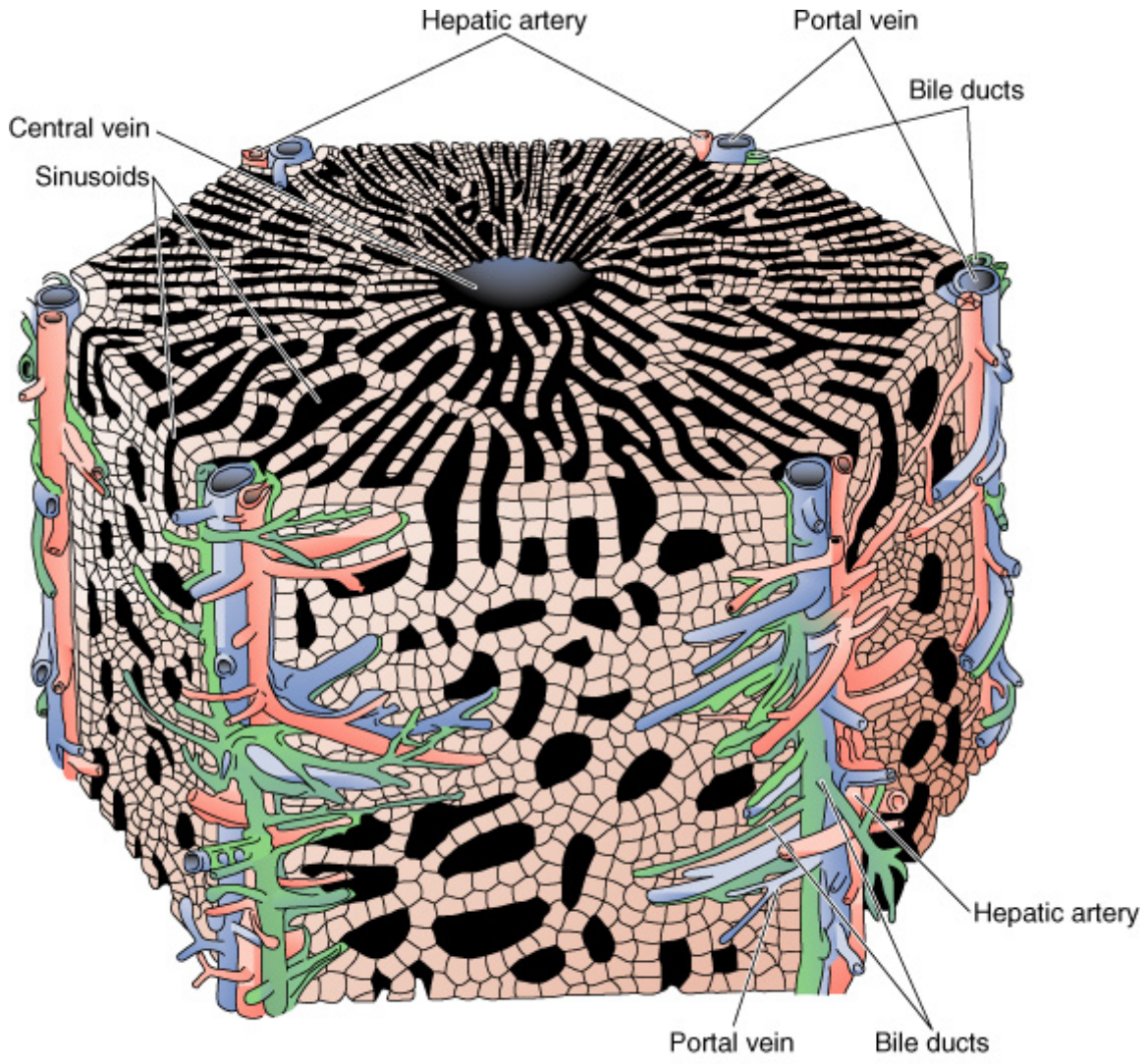


A

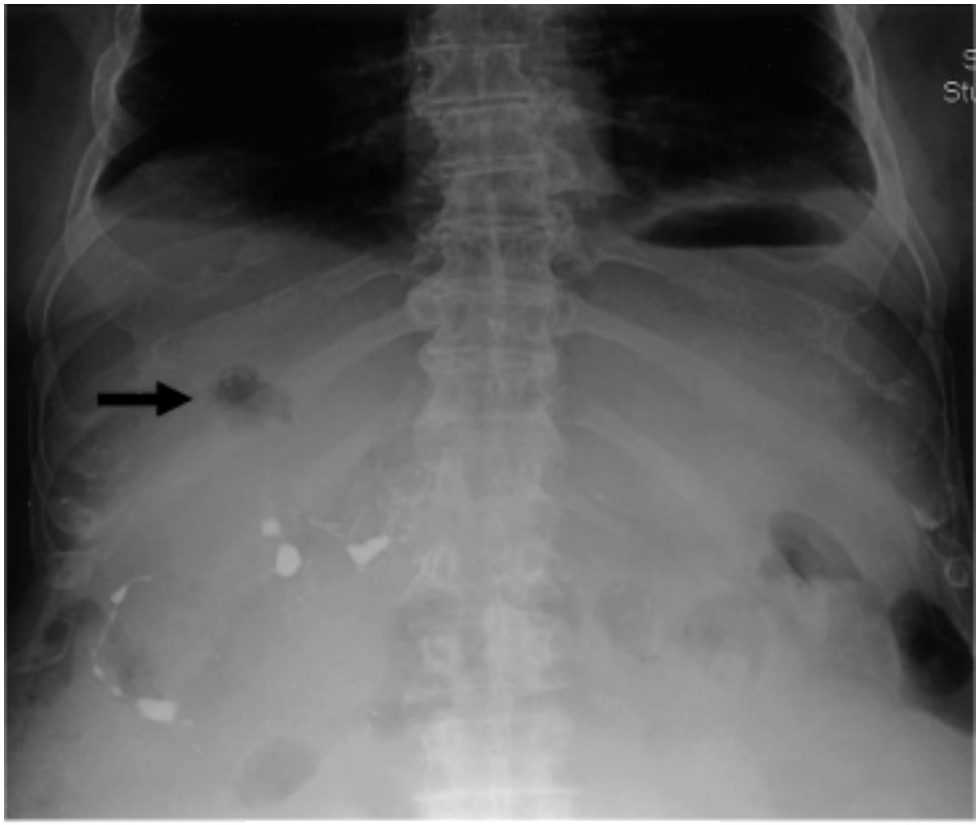


B

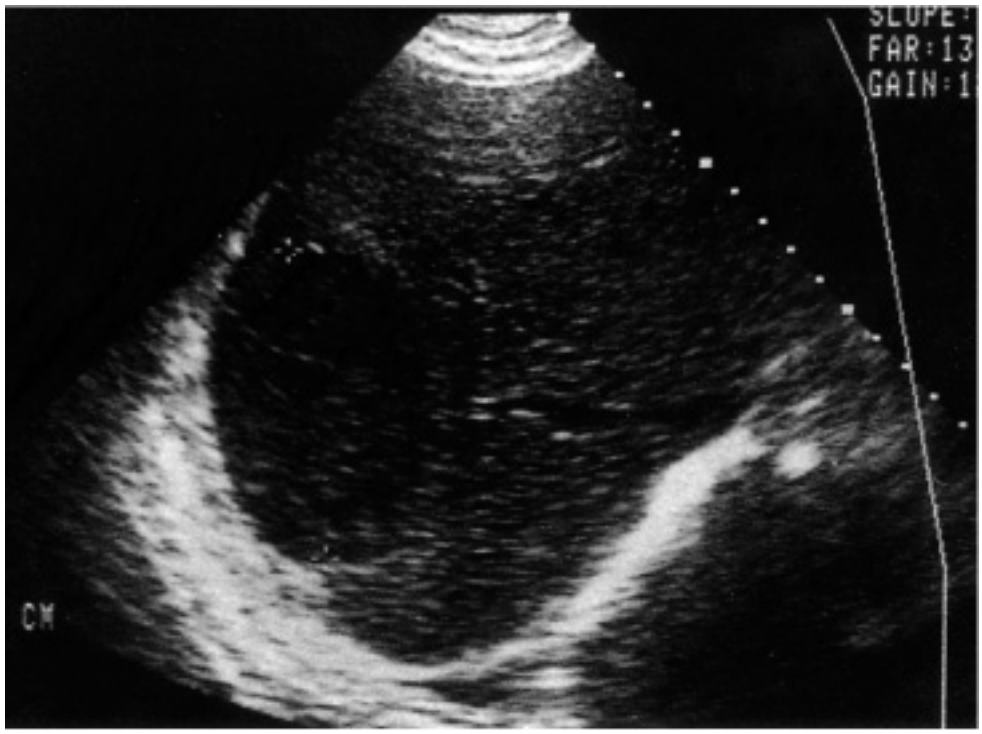
SEGMENTAL ANATOMY OF LIVER



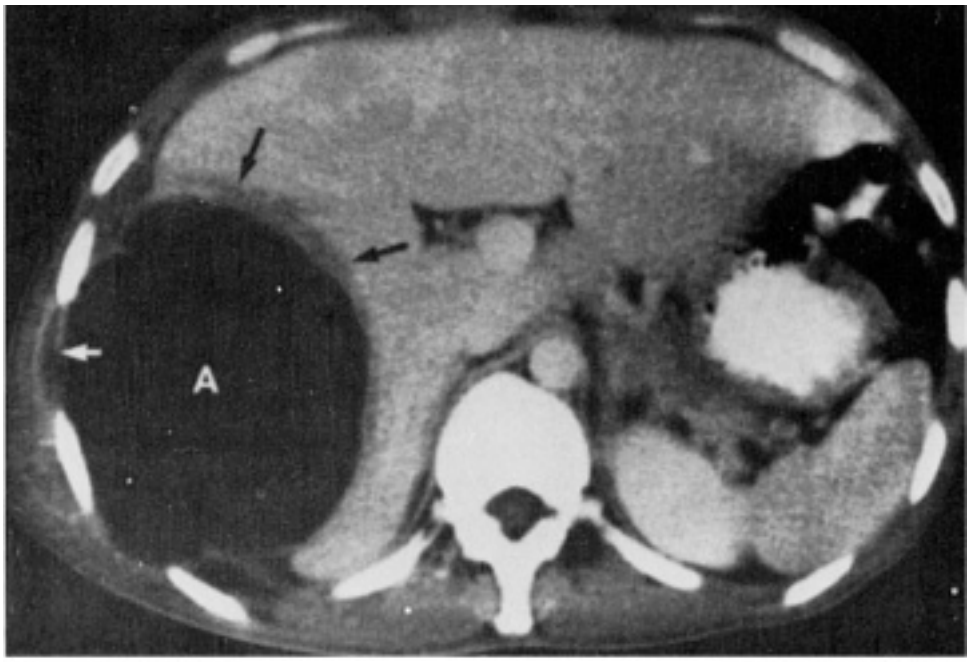
HEPATIC LOBULE

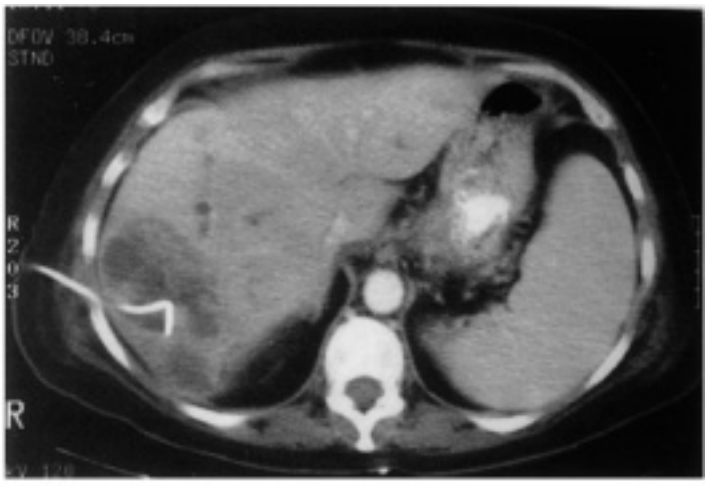


LIVER ABSCESS WITH FLUID LEVEL INSIDE ABSCESS CAVITY



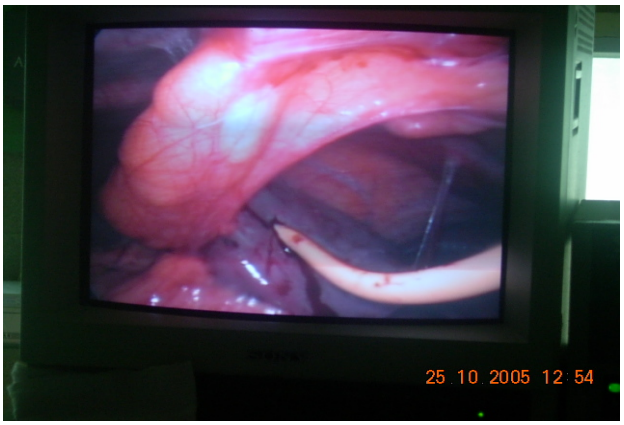
RT LOBE ABSCESS

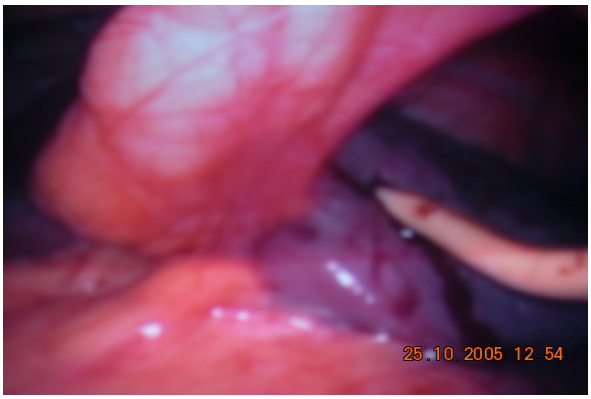




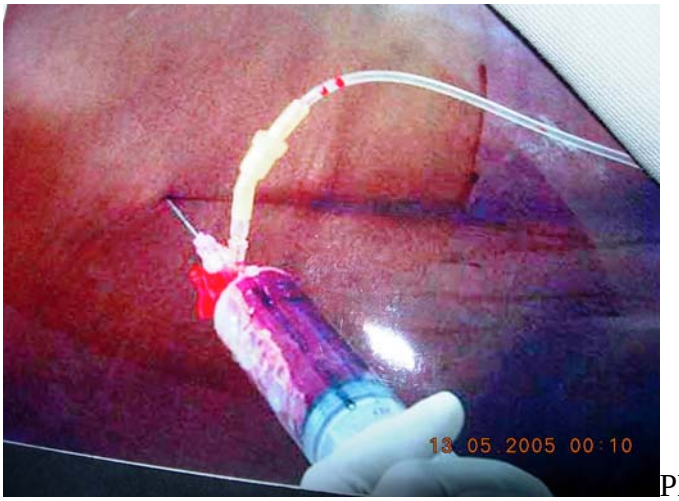
B

LARGE RT LOBE ABSCESS AND PERCUTANEOUS CATHETER DRAINAGE



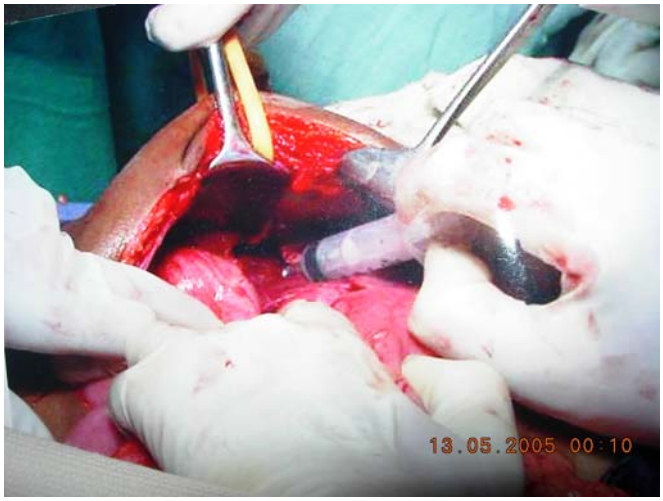


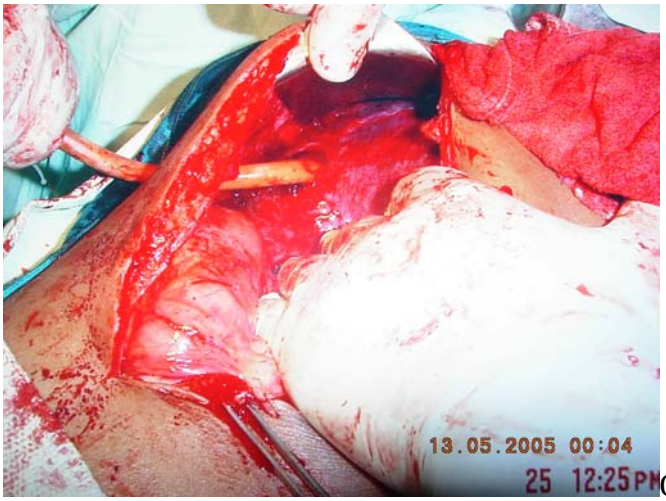
LAPROSCOPIC DRAINAGE



PERCUTANEOUS ASPIRATION

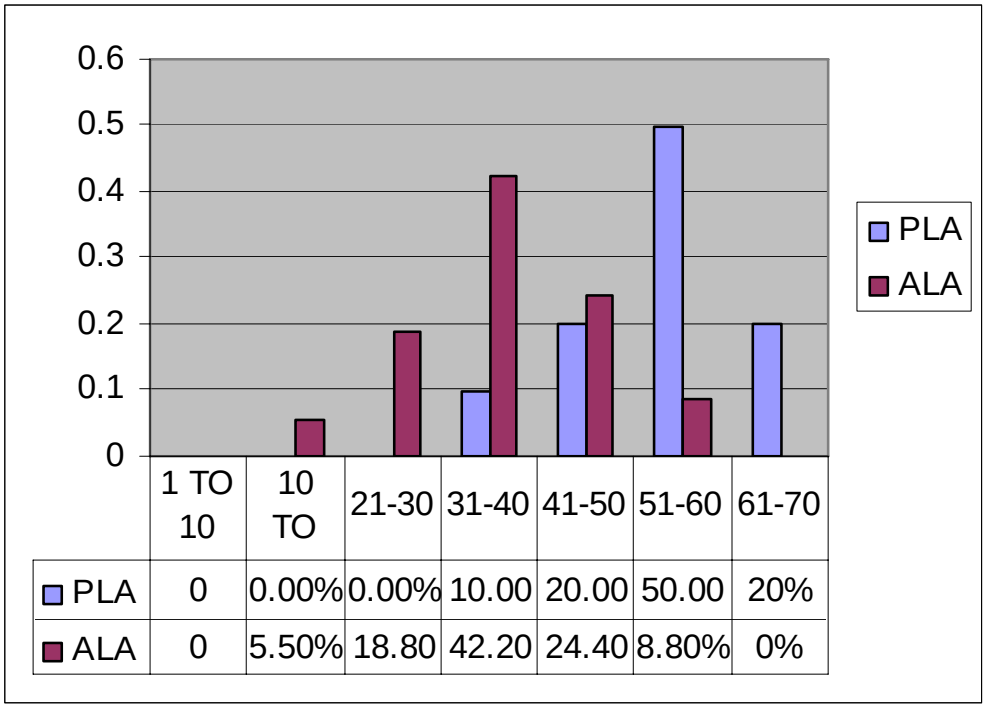
PERCUTANEOUS CATHETER DRAINAGE





OPEN SURGICAL DRAINAGE

AGE DISTRIBUTION



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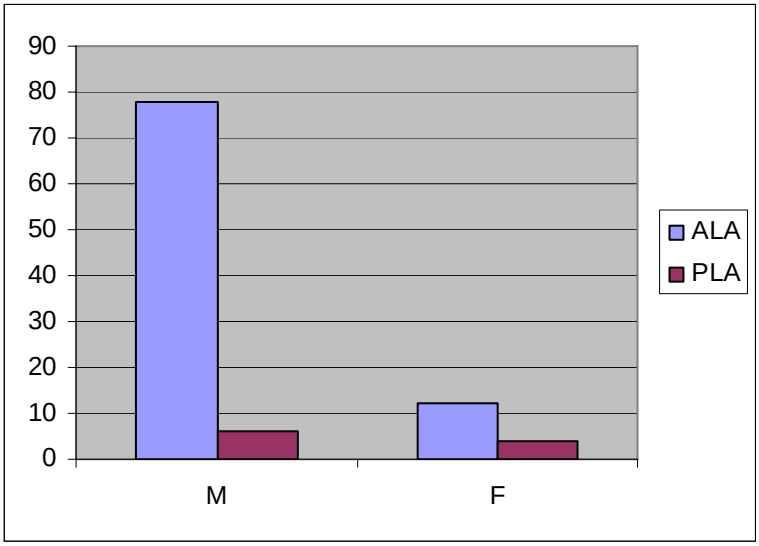
ALA	0	5.50%	18.80	42.20	24.40	8.80%	0%

MALE:FEMALE DISTRIBUTION



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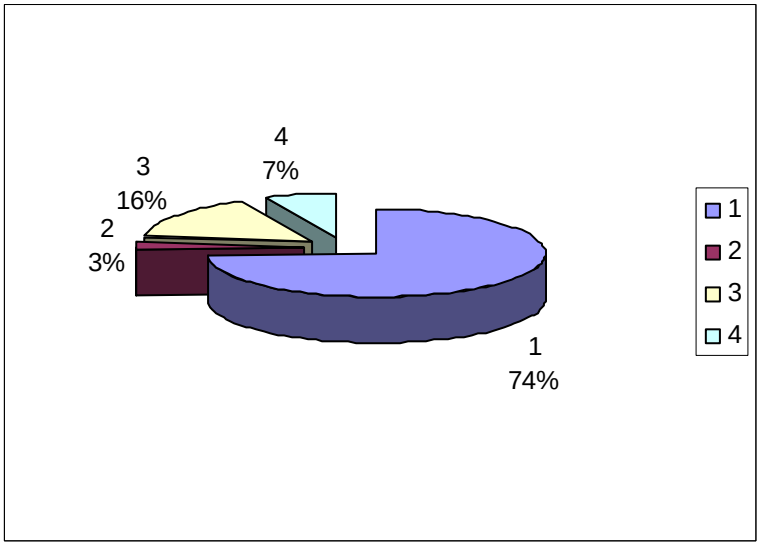
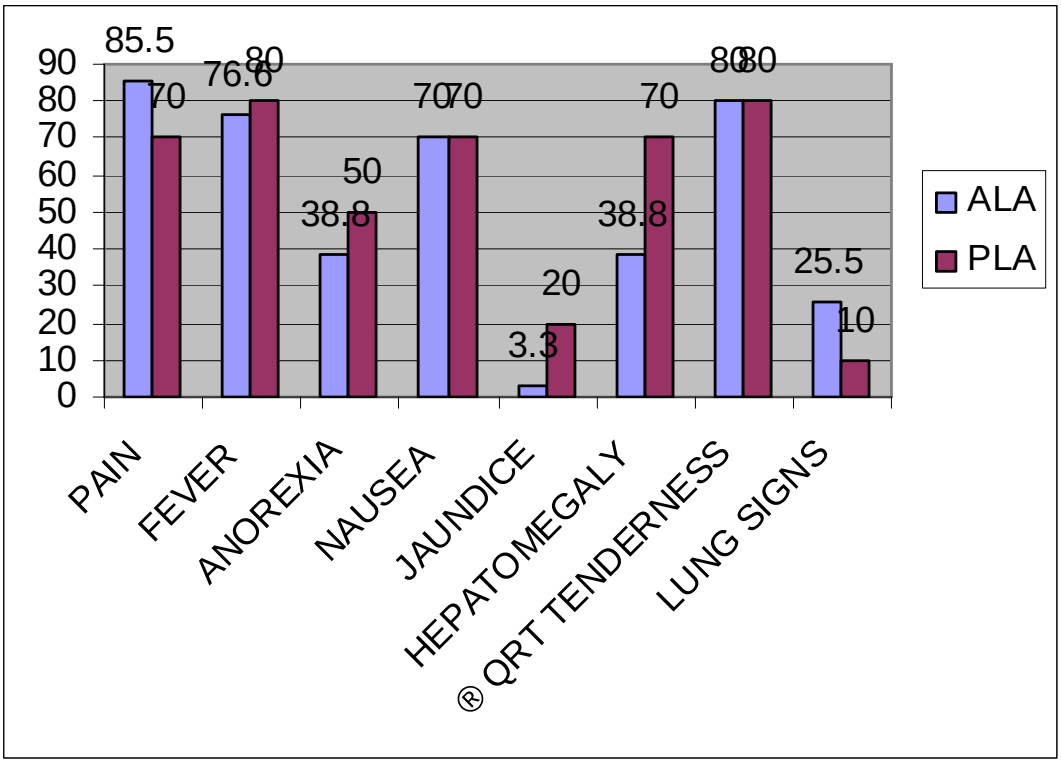
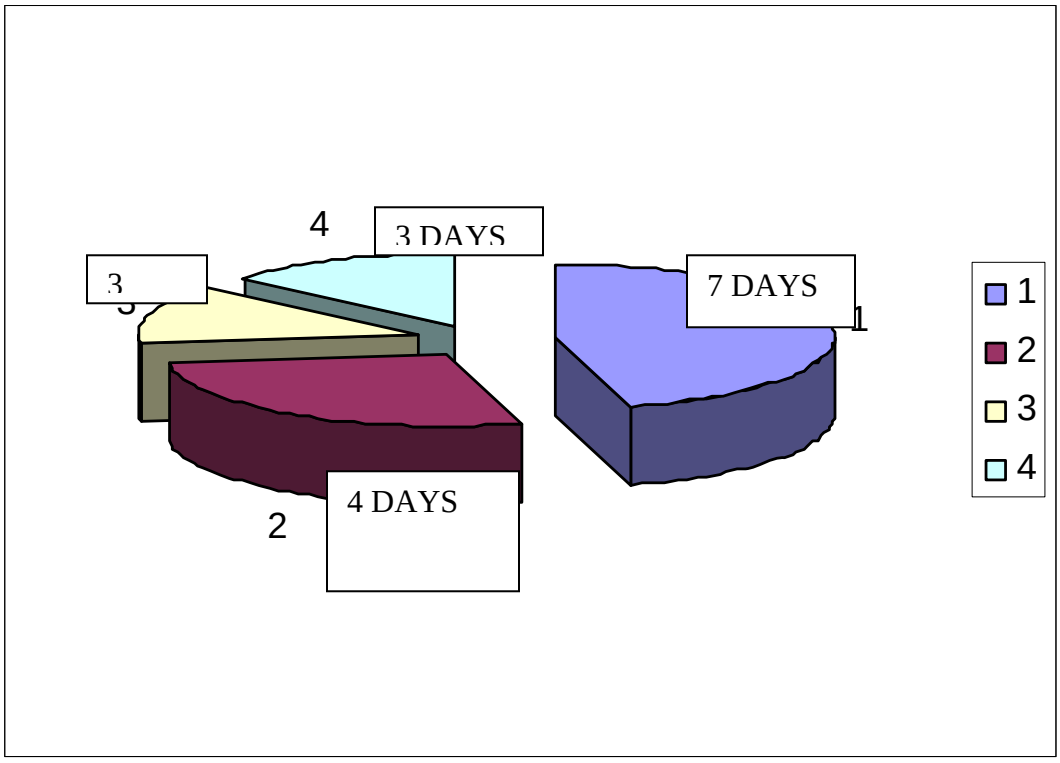


SYMPTOM AND SIGN ANALYSIS



TREATMENT ANALYSIS



sno	Name	Age/ sex	Ip number	pain	fever	anorexia	hepatom	Rt hpo	Lung	jaundice	Lobe	diagnosis	treatment
1.	Arunpandy	28/m	465783	+	+	+		+	+		R	ala	Medical
2	Selvaraj	27/m	476382	+	+		+	+			L	ala	Medical
3	Rathibeevi	65/f	487654	+	+			+			R	pla	pcd
4	Muthu	21/m	365478	+	+		+				R	ala	pca
5	Bose	48/m	387654	+			+	+			R	ala	pca
6	Rajan	38/m	459862	+	+	+		+	+		R	ala	pca
7	Veeranan	50/m	348760	+	+	+		+	+		R	ala	pca
8	Chinakal	39/m	459876	+	+	+	+	+			R	ala	pca
9	Murugesa	35/m	325678	+		+		+			B	ala	medical
10	M.ali jinnah	22/m	412376	+	+			+			R	ala	pca
11	Kasidevar	50/m	326574	+	+	+		+			B	pla	medical
12	Ravi	30/m	465732	+	+		+	+			R	ala	medical
13	Annoniammal	50/f	437824	+	+			+			B	pla	medical
14	Nagaraj	36/m	327650	+	+	+	+	+			B	ala	pcd
15	Selvan	35/m	412385		+	+		+	+		R	ala	medical
16	Pandian	40/m	431275	+	+			+			R	ala	pca
17	Mariappan	35/m	367496	+	+			+			L	ala	pca
18	Amran	38/m	327498	+	+	+		+			R	ala	pca
19	Subulakshmi	47/f	356537	+	+	+			+		R	ala	pca
20	Angeshwaran	35/m	476518	+	+		+	+			R	pla	pca
21	Ramanan	16/m	450956	+	+		+	+			L	ala	Lcd

sno	name	Age/ sex	Ip number	pain	fever	anorexia	hepatom	Rt hypo	lung	jaundice	lobe	diagnosis	treatment
22.	Balasubramanian	37/m	327814	+	+		+	+			R	ala	pca
23.	Selvaraj	33/m	423716	+	+	+		+			R	ala	pca
24.	Karuppiah	29/m	400780	+	+			+			B	ala	medical
25	Alukkan	32/m	326589		+	+	+	+		+	B	pla	medical
26	Pappammal	55/f	300786	+	+						L	ala	pca
27	Ashok	32/m	211657	+				+			B	ala	pca
28	Sulaiman	50/m	214698	+	+		+	+			R	ala	pca
29	Vellaisamy	20/m	376260	+	+	+					B	ala	medical
30	Ratchagan	50/m	314287	+		+	+		+		R	ala	pca
31	Muthuraj	46/m	243567	+		+		+			R	ala	pca
32	Vellaiammal	45/f	209743	+			+	+	+		R	pla	pca
33	Lakshmanan	33/m	421567	+	+			+			R	ala	Pca
34	Karuppiah	50/m	265738	+	+	+	+				R	pla	medical
35	Chandran	32/m	413847	+	+			+			R	ala	medical
36	Nagappan	30/m	210624	+	+						L	ala	medical
37	Subbiah	45/m	398333	+	+	+	+	+	+		B	ala	medical
38	Ganesan	40/m	412998	+			+	+	+		R	ala	pca
39	Raju	25/m	333657	+	+		+	+		+	B	ala	pca
40	Ayyapan	27/m	408076	+	+						R	ala	medical
41	Vincent	31/m	243856	+		+		+	+		R	ala	pca
42	Rathinavel	40/m	289004	+	+			+	+		R	ala	pca
43	Madasamy	35/m	311517	+	+			+			R	ala	pcd
44	Ramamoorthy	35/m	395176	+	+		+	+			R	ala	pca
45	Rajaguru	30/m	476139	+	+			+			L	ala	Lcd
46	Raju	28/m	444915		+	+		+			R	ala	medical
47	Perumalsamy	46/m	423857		+	+	+	+	+		R	ala	pca
48	Muthuvel	42/m	311569	+	+		+		+		R	ala	pca
49	Alagarsamy	37/m	411712			+	+				R	ala	medical
50	Gurusamy	36/m	442177	+	+			+			R	ala	pcd
51	Aivan	25/m	344456	+	+				+		L	ala	medical
52	Mayalugu	45/m	276009	+		+		+			L	ala	pca
53	Mottayan	38/m	266775	+	+		+	+			B	ala	pca
54	Dhanam	46/m	255664	+	+			+			R	ala	pca
55	Ramayee	53/f	233445		+	+	+	+		+	B	pla	pca
56	Veluthevar	60/m	477513	+				+			R	ala	medical
57	Mokkai	30/m	211874	+	+		+	+			L	ala	Lcd
58	Paul	30/m	300587	+	+			+			R	ala	pca
59	Asaimalai	45/m	273356		+		+	+			B	pla	medical
60	Pitchaiammal	42/f	264985	+				+			R	Ala	pca

Sno	Name	Age/ sex	Ip number	pain	fever	anorexia	hepatom egaly	Rt hypo tend	jaundice	Lung signs	lobe	diadnosis	treatment
61	Ravi	13/m	225543	+	+			+			R	ala	medical
62	Palanisamy	37/m	417758	+			+	+			R	ala	pca
63	Ramasamy	32/m	266498		+	+		+			R	ala	medical
64	Ramachandran	38/m	367345	+		+	+			+	R	ala	pca
65	Dhanushkodi	26/m	477881	+	+			+			R	ala	pca
66	Jeyasudha	27/f	345198	+	+		+	+	+		R	ala	pcd
67	Mani	35/f	418854		+			+			R	ala	medical
68	Thiruchulian	40/m	270714	+	+		+				L	ala	pca
69	Mari	40/m	300678	+		+		+			R	ala	medical
70	Chandra	36/f	201020	+		+			+	+	R	ala	pca
71	Saravanan	30/m	403030		+	+					R	ala	medical
72	Chinnan	35/m	230954	+	+			+			R	ala	pca
73	Vallaisamy	50/m	253635	+	+		+	+		+	R	ala	pca
74	Palaniappan	48/m	367109	+			+	+			R	ala	pca
75	Kumaravel	54/m	367480		+	+	+			+	R	ala	pca
76	Mariammal	38/f	253987	+				+			R	ala	pca
77	Narayanan	51/m	310440	+	+			+			B	ala	medical
78	Balusamy	36/m	411669		+		+	+			R	Ala	pcd
79	Karupiah	25/m	442694	+	+		+	+		+	R	ala	pcd
80	Sethuraman	18/m	476289	+		+		+			R	ala	pca
81	Kumaran	36/m	400996	+	+	+		+		+	R	ala	pcd
82	Renganathan	41/m	430900	+	+	+	+	+		+	R	ala	pcd
83	Jeyamani	36/f	300108	+	+			+			R	ala	Lcd
84	Raman	49/m	298456		+	+		+			B	ala	pcd
85	Muthu	37/m	288780	+	+	+		+			R	ala	pcd
86	Muthulaksmi	45/f	444332	+	+			+			R	ala	pca
87	Anandhi	33/f	413286		+	+	+				R	ala	pca
88	Anitha	38/f	411312	+	+			+		+	R	ala	Lcd
89	Krishnan	31/m	419996		+	+	+				L	ala	pca
90	Mumtaj	22/f	234516	+		+		+			R	ala	pca
91	Ravi	37/m	411332	+	+						R	ala	medical
92	Moorthy	46/m	294445	+	+		+	+		+	R	ala	pca
93	Muniappan	47/m	211338	+		+		+			R	ala	medical
94	Mayan	45/m	411689	+	+			+			R	ala	pca
95	Rajangam	41/m	315125	+		+	+	+			R	ala	pca
96	Ramar	16/m	467421	+	+		+	+			L	ala	Lcd
97	Kuppan	35/m	213911	+	+			+			B	ala	medical
98	Devika	28/m	416171	+	+		+	+			R	ala	Lcd
99	Murugesan	40/m	214657		+		+			+	R	ala	Lcd
100	Ramachandran	40/m	314703	+		+		+			L	pla	Medical